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```
=> s nocel approach to specific growth factor
    0 NOCEL
    509480 APPROACH
    169969 APPROACHES
    643384 APPROACH
        (APPROACH OR APPROACHES)
    10075147 TO
        1074 TOS
    10075444 TO
        (TO OR TOS)
    1246737 SPECIFIC
        1438 SPECIFICS
    1247887 SPECIFIC
        (SPECIFIC OR SPECIFICS)
    1027883 GROWTH
        1922 GROWTHS
    1029285 GROWTH
        (GROWTH OR GROWTHS)
    953627 FACTOR
    2381874 FACTORS
    2979682 FACTOR
        (FACTOR OR FACTORS)
L1      0 NOCEL APPROACH TO SPECIFIC GROWTH FACTOR
        (NOCEL(W)APPROACH(W)TO(W)SPECIFIC(W)GROWTH(W)FACTOR)

=> s novel approach to specific growth factor
    405621 NOVEL
        273 NOVELS
    405846 NOVEL
        (NOVEL OR NOVELS)
    509480 APPROACH
    169969 APPROACHES
    643384 APPROACH
        (APPROACH OR APPROACHES)
    10075147 TO
        1074 TOS
    10075444 TO
        (TO OR TOS)
    1246737 SPECIFIC
        1438 SPECIFICS
    1247887 SPECIFIC
        (SPECIFIC OR SPECIFICS)
    1027883 GROWTH
        1922 GROWTHS
    1029285 GROWTH
        (GROWTH OR GROWTHS)
    953627 FACTOR
    2381874 FACTORS
    2979682 FACTOR
        (FACTOR OR FACTORS)
L2      1 NOVEL APPROACH TO SPECIFIC GROWTH FACTOR
        (NOVEL(W)APPROACH(W)TO(W)SPECIFIC(W)GROWTH(W)FACTOR)
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=> d bib ab

L2 ANSWER 1 OF 1 MEDLINE on STN  
 AN 1999113969 MEDLINE <<LOGINID::20090818>>  
 DN PubMed ID: 9916931  
 TI Novel approach to specific  
     growth factor inhibition *in vivo*: antagonism of  
     platelet-derived growth factor in glomerulonephritis by aptamers.  
 AU Floege J; Ostendorf T; Janssen U; Burg M; Radeke H H; Vargeese C; Gill S  
     C; Green L S; Janjic N  
 CS Division of Nephrology, Medizinische Hochschule, Hannover, Germany..  
     Floege.Juergen@MH-Hannover.de  
 SO The American journal of pathology, (1999 Jan) Vol. 154, No. 1, pp. 169-79.  
     Journal code: 0370502. ISSN: 0002-9440.  
     Report No.: NLM-PMC1853442.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
     (RESERCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Abridged Index Medicus Journals; Priority Journals  
 EM 199902  
 ED Entered STN: 23 Feb 1999  
     Last Updated on STN: 3 Mar 2000  
     Entered Medline: 11 Feb 1999  
 AB Mesangial cell proliferation and matrix accumulation, driven by platelet-derived growth factor (PDGF), contribute to many progressive renal diseases. In a novel approach to antagonize PDGF, we investigated the effects of a nuclease-resistant high-affinity oligonucleotide aptamer *in vitro* and *in vivo*. In cultured mesangial cells, the aptamer markedly suppressed PDGF-BB but not epidermal- or fibroblast-growth-factor-2-induced proliferation. *In vivo* effects of the aptamer were evaluated in a rat mesangioproliferative glomerulonephritis model. Twice-daily intravenous (i.v.) injections from days 3 to 8 after disease induction of 2.2 mg/kg PDGF-B aptamer, coupled to 40-kd polyethylene glycol (PEG), led to 1) a reduction of glomerular mitoses by 64% on day 6 and by 78% on day 9, 2) a reduction of proliferating mesangial cells by 95% on day 9, 3) markedly reduced glomerular expression of endogenous PDGF B-chain, 4) reduced glomerular monocyte/macrophage influx on day 6 after disease induction, and 5) a marked reduction of glomerular extracellular matrix overproduction (as assessed by analysis of fibronectin and type IV collagen) both on the protein and mRNA level. The administration of equivalent amounts of a PEG-coupled aptamer with a scrambled sequence or PEG alone had no beneficial effect on the natural course of the disease. These data show that specific inhibition of growth factors using custom-designed, high-affinity aptamers is feasible and effective.

=> FIL STNGUIDE  
 COST IN U.S. DOLLARS  
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NEWS 3 JUN 01 CAS REGISTRY Source of Registration (SR) searching enhanced on STN  
NEWS 4 JUN 26 NUTRACEUT and PHARMAML no longer updated  
NEWS 5 JUN 29 IMSCOPROFILE now reloaded monthly  
NEWS 6 JUN 29 EPFULL adds Simultaneous Left and Right Truncation (SLART) to AB, MCLM, and TI fields  
NEWS 7 JUL 09 PATDPAFULL adds Simultaneous Left and Right Truncation (SLART) to AB, CLM, MCLM, and TI fields  
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NEWS 9 JUL 27 CA/CAplus enhanced with new citing references  
NEWS 10 JUL 16 GBFULL adds patent backfile data to 1855  
NEWS 11 JUL 21 USGENE adds bibliographic and sequence information  
NEWS 12 JUL 28 EPFULL adds first-page images and applicant-cited references  
NEWS 13 JUL 28 INPADOCDB and INPAFAMDB add Russian legal status data  
NEWS 14 AUG 08 Improve STN by completing a survey and be entered to win a gift card  
NEWS 15 AUG 10 Time limit for inactive STN sessions doubles to 40 minutes  
NEWS 16 AUG 17 CAS REGISTRY, the Global Standard for Chemical Research, Approaches 50 Millionth Registration Milestone  
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* http://www.zoomerang.com/Survey/?p=WEB229H4S8Q5UL *  
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FILE 'HOME' ENTERED AT 07:14:07 ON 20 AUG 2009

FILE 'MEDLINE' ENTERED AT 07:14:20 ON 20 AUG 2009

FILE LAST UPDATED: 19 Aug 2009 (20090819/UP). FILE COVERS 1949 TO DATE.

MEDLINE and LMEDLINE have been updated with the 2009 Medical Subject Headings (MeSH) vocabulary and tree numbers from the U.S. National Library of Medicine (NLM). Additional information is available at

[http://www.nlm.nih.gov/pubs/techbull/nd08/nd08\\_medline\\_data\\_changes\\_2009.html](http://www.nlm.nih.gov/pubs/techbull/nd08/nd08_medline_data_changes_2009.html).

On February 21, 2009, MEDLINE was reloaded. See HELP RLOAD for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

See HELP RANGE before carrying out any RANGE search.

```
=> s mismatch? and sirna  
      26652 MISMATCH?  
      10822 SIRNA  
      2680  SIRNAS  
      11832 SIRNA  
                           (SIRNA OR SIRNAS)  
L1          117  MISMATCH? AND SIRNA
```

=> d ti 70-117

L1 ANSWER 70 OF 117 MEDLINE on STN  
TI Passenger-strand cleavage facilitates assembly of siRNA into Ago2-containing RNAi enzyme complexes.

L1 ANSWER 71 OF 117 MEDLINE on STN  
TI An efficient intrathecal delivery of small interfering RNA to the spinal cord and peripheral neurons.

L1 ANSWER 72 OF 117 MEDLINE on STN  
TI 518A2 melanoma cells are protected by G3139 and other antineoplastic agents against the cytotoxic effects of DTIC.

L1 ANSWER 73 OF 117 MEDLINE on STN  
TI Biomembrane-permeable and Ribonuclease-resistant siRNA with enhanced activity.

L1 ANSWER 74 OF 117 MEDLINE on STN  
TI Quantum dot-conjugated hybridization probes for preliminary screening of siRNA sequences.

L1 ANSWER 75 OF 117 MEDLINE on STN  
TI Differential nonsense mediated decay of mutated mRNAs in mismatch repair deficient colorectal cancers.

L1 ANSWER 76 OF 117 MEDLINE on STN  
TI The role RNA interference of alpha<sub>1</sub>, 3GT plays in resistance to complement mediated cytotoxicity of porcine endothelial cells.

L1 ANSWER 77 OF 117 MEDLINE on STN  
TI Specific effects of microRNAs on the plant transcriptome.

L1 ANSWER 78 OF 117 MEDLINE on STN  
TI Inhibition of vascular endothelial growth factor gene expression by T7-siRNAs in cultured human retinal pigment epithelial cells.

L1 ANSWER 79 OF 117 MEDLINE on STN  
TI Accelerated off-target search algorithm for siRNA.

L1 ANSWER 80 OF 117 MEDLINE on STN  
TI hMutS alpha is protected from ubiquitin-proteasome-dependent degradation by atypical protein kinase C zeta phosphorylation.

L1 ANSWER 81 OF 117 MEDLINE on STN  
TI A computational study of off-target effects of RNA interference.

L1 ANSWER 82 OF 117 MEDLINE on STN  
TI Silencing of human c-myc oncogene expression by poly-DNP-RNA.

L1 ANSWER 83 OF 117 MEDLINE on STN  
TI Osteopontin silencing by small interfering RNA suppresses in vitro and in vivo CT26 murine colon adenocarcinoma metastasis.

L1 ANSWER 84 OF 117 MEDLINE on STN  
TI A systematic analysis of the silencing effects of an active siRNA at all single-nucleotide mismatched target sites.

L1 ANSWER 85 OF 117 MEDLINE on STN  
TI Evaluation of in vitro inhibitory potential of small interfering RNAs directed against various regions of foot-and-mouth disease virus genome.

L1 ANSWER 86 OF 117 MEDLINE on STN  
TI Influence of assembly of siRNA elements into RNA-induced silencing complex by fork-siRNA duplex carrying nucleotide mismatches at the 3'- or 5'-end of the sense-stranded siRNA element.

L1 ANSWER 87 OF 117 MEDLINE on STN  
TI Inhibition of coxsackievirus B3 replication by small interfering RNAs requires perfect sequence match in the central region of the viral positive strand.

L1 ANSWER 88 OF 117 MEDLINE on STN

TI ESPSearch: a program for finding exact sequences and patterns in DNA, RNA, or protein.

L1 ANSWER 89 OF 117 MEDLINE on STN

TI Poliovirus escape from RNA interference: short interfering RNA-target recognition and implications for therapeutic approaches.

L1 ANSWER 90 OF 117 MEDLINE on STN

TI Rescue of the TTF2 knockdown phenotype with an siRNA-resistant replacement vector.

L1 ANSWER 91 OF 117 MEDLINE on STN

TI HPC1/RNASEL mediates apoptosis of prostate cancer cells treated with 2',5'-oligoadenylates, topoisomerase I inhibitors, and tumor necrosis factor-related apoptosis-inducing ligand.

L1 ANSWER 92 OF 117 MEDLINE on STN

TI Validating siRNA using a reporter made from synthetic DNA oligonucleotides.

L1 ANSWER 93 OF 117 MEDLINE on STN

TI Cloning and gene silencing of LAT2, the L-3,4-dihydroxyphenylalanine (L-DOPA) transporter, in pig renal LLC-PK1 epithelial cells.

L1 ANSWER 94 OF 117 MEDLINE on STN

TI Systemic siRNA-mediated gene silencing: a new approach to targeted therapy of cancer.

L1 ANSWER 95 OF 117 MEDLINE on STN

TI Picking a winner: new mechanistic insights into the design of effective siRNAs.

L1 ANSWER 96 OF 117 MEDLINE on STN

TI Retrovirally mediated RNA interference targeting the M2 subunit of ribonucleotide reductase: A novel therapeutic strategy in pancreatic cancer.

L1 ANSWER 97 OF 117 MEDLINE on STN

TI Use of siRNAs and antisense oligonucleotides against survivin RNA to inhibit steps leading to tumor angiogenesis.

L1 ANSWER 98 OF 117 MEDLINE on STN

TI Poly-2'-DNP-RNAs with enhanced efficacy for inhibiting cancer cell growth.

L1 ANSWER 99 OF 117 MEDLINE on STN

TI Downregulation of Bcl-2, FLIP or IAPs (XIAP and survivin) by siRNAs sensitizes resistant melanoma cells to Apo2L/TRAIL-induced apoptosis.

L1 ANSWER 100 OF 117 MEDLINE on STN

TI Many commonly used siRNAs risk off-target activity.

L1 ANSWER 101 OF 117 MEDLINE on STN

TI RISC is a 5' phosphomonoester-producing RNA endonuclease.

L1 ANSWER 102 OF 117 MEDLINE on STN

TI Mismatch repair-mediated G2/M arrest by 6-thioguanine involves the ATR-Chk1 pathway.

L1 ANSWER 103 OF 117 MEDLINE on STN

TI Derivation and function of small interfering RNAs and microRNAs.

L1 ANSWER 104 OF 117 MEDLINE on STN  
TI Nucleotide-based therapies targeting clusterin chemosensitize human lung adenocarcinoma cells both in vitro and in vivo.

L1 ANSWER 105 OF 117 MEDLINE on STN  
TI Differentiated human podocytes endogenously express an inhibitory isoform of vascular endothelial growth factor (VEGF165b) mRNA and protein.

L1 ANSWER 106 OF 117 MEDLINE on STN  
TI Enhancement of RNAi activity by improved siRNA duplexes.

L1 ANSWER 107 OF 117 MEDLINE on STN  
TI The p38 mitogen-activated protein kinase pathway links the DNA mismatch repair system to the G2 checkpoint and to resistance to chemotherapeutic DNA-methylating agents.

L1 ANSWER 108 OF 117 MEDLINE on STN  
TI Functional siRNAs and miRNAs exhibit strand bias.

L1 ANSWER 109 OF 117 MEDLINE on STN  
TI Asymmetry in the assembly of the RNAi enzyme complex.

L1 ANSWER 110 OF 117 MEDLINE on STN  
TI Specific gene silencing using small interfering RNAs in fish embryos.

L1 ANSWER 111 OF 117 MEDLINE on STN  
TI Allele-specific silencing of a pathogenic mutant acetylcholine receptor subunit by RNA interference.

L1 ANSWER 112 OF 117 MEDLINE on STN  
TI siRNA function in RNAi: a chemical modification analysis.

L1 ANSWER 113 OF 117 MEDLINE on STN  
TI Efficient reduction of target RNAs by small interfering RNA and RNase H-dependent antisense agents. A comparative analysis.

L1 ANSWER 114 OF 117 MEDLINE on STN  
TI Effects on RNA interference in gene expression (RNAi) in cultured mammalian cells of mismatches and the introduction of chemical modifications at the 3'-ends of siRNAs.

L1 ANSWER 115 OF 117 MEDLINE on STN  
TI RNA interference by expression of short-interfering RNAs and hairpin RNAs in mammalian cells.

L1 ANSWER 116 OF 117 MEDLINE on STN  
TI Positional effects of short interfering RNAs targeting the human coagulation trigger Tissue Factor.

L1 ANSWER 117 OF 117 MEDLINE on STN  
TI Functional anatomy of siRNAs for mediating efficient RNAi in Drosophila melanogaster embryo lysate.

=> d bib ab 114 116 112 111 106 95 86 85 84

L1 ANSWER 114 OF 117 MEDLINE on STN  
AN 2002714933 MEDLINE <<LOGINID::20090820>>  
DN PubMed ID: 12477280  
TI Effects on RNA interference in gene expression (RNAi) in cultured

mammalian cells of mismatches and the introduction of chemical modifications at the 3'-ends of siRNAs.

AU Hamada Makiko; Ohtsuka Toshiaki; Kawaida Reimi; Koizumi Makoto; Morita Koji; Furukawa Hidehiko; Imanishi Takeshi; Miyagishi Makoto; Taira Kazunari

CS Biomedical Research Laboratories, Sankyo Co., Ltd., Tokyo 140-8710, Japan.

SO Antisense & nucleic acid drug development, (2002 Oct) Vol. 12, No. 5, pp. 301-9.

Journal code: 9606142. ISSN: 1087-2906.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200305

ED Entered STN: 17 Dec 2002  
Last Updated on STN: 9 May 2003  
Entered Medline: 8 May 2003

AB The highly specific posttranscriptional silencing of gene expression induced by double-stranded RNA (dsRNA) is known as RNA interference (RNAi) and has been demonstrated in plants, nematodes, Drosophila, and protozoa, as well as in mammalian cells. The suppression of expression of specific genes by chemically synthesized 21-nucleotide (21-nt) RNA duplexes has been achieved in various lines of mammalian cells, and this technique might prove to be a valuable tool in efforts to analyze biologic functions of genes in mammalian cells. In order to investigate the utility of potential modifications that can be introduced into small interfering RNAs (siRNAs) and also to study their functional anatomy, we synthesized different types of siRNA targeted to mRNA of Jun dimerization protein 2 (JDP2). Our detailed analysis demonstrated that siRNAs with only one mismatch, relative to the target, on the antisense strand had reduced RNAi effect, whereas the corresponding mutation on the sense strand did not interfere with the RNAi. Moreover, one 2-hydroxyethylphosphate (hp) substitution at the 3'-end of the antisense strand but not of the sense strand also prevented RNAi, whereas a related modification at the 3'-end of either strand, using 2'-O,4'-C-ethylene thymidine (eT), which is a component of ethylene-bridge nucleic acids (ENA), completely abolished RNAi. These results support the hypothesis that the two strands have different functions in RNAi in cultured mammalian cells and indicate that their chemical modification of siRNAs at the 3'-end of the sense strand exclusively is possible, without loss of RNAi activity, depending on the type of modification. Because modification at the 3'-end of the antisense strand by hp or eT abolished the RNAi effect, it appears possible that the 3'-end is recognized by the RNA-induced silencing complex (RISC).

L1 ANSWER 116 OF 117 MEDLINE on STN  
AN 2002204990 MEDLINE <<LOGINID::20090820>>  
DN PubMed ID: 11937629

TI Positional effects of short interfering RNAs targeting the human coagulation trigger Tissue Factor.

AU Holen Torgeir; Amarzguioui Mohammed; Wiiger Merete T; Babaie Eshrat; Prydz Hans

CS The Biotechnology Centre of Oslo, University of Oslo, Gaustadalleen 21, N-0349 Oslo, Norway.

SO Nucleic acids research, (2002 Apr 15) Vol. 30, No. 8, pp. 1757-66.  
Journal code: 0411011. E-ISSN: 1362-4962.  
Report No.: NLM-PMC113209.

CY England: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English

FS Priority Journals  
EM 200204  
ED Entered STN: 9 Apr 2002  
Last Updated on STN: 19 Apr 2002  
Entered Medline: 18 Apr 2002  
AB Chemically synthesised 21-23 bp double-stranded short interfering RNAs ( siRNA) can induce sequence-specific post-transcriptional gene silencing, in a process termed RNA interference (RNAi). In the present study, several siRNAs synthesised against different sites on the same target mRNA (human Tissue Factor) demonstrated striking differences in silencing efficiency. Only a few of the siRNAs resulted in a significant reduction in expression, suggesting that accessible siRNA target sites may be rare in some human mRNAs. Blocking of the 3'-OH with FITC did not reduce the effect on target mRNA. Mutations in the siRNAs relative to target mRNA sequence gradually reduced, but did not abolish mRNA depletion. Inactive siRNAs competed reversibly with active siRNAs in a sequence-independent manner. Several lines of evidence suggest the existence of a near equilibrium kinetic balance between mRNA production and siRNA-mediated mRNA depletion. The silencing effect was transient, with the level of mRNA recovering fully within 4-5 days, suggesting absence of a propagative system for RNAi in humans. Finally, we observed 3' mRNA cleavage fragments resulting from the action of the most effective siRNAs. The depletion rate-dependent appearance of these fragments argues for the existence of a two-step mRNA degradation mechanism.

L1 ANSWER 112 OF 117 MEDLINE on STN  
AN 2003437660 MEDLINE <<LOGINID::20090820>>  
DN PubMed ID: 12923253  
TI siRNA function in RNAi: a chemical modification analysis.  
AU Chiu Ya-Lin; Rana Tariq M  
CS Department of Biochemistry and Molecular Pharmacology, University of Massachusetts Medical School, Worcester, Massachusetts 01605, USA.  
NC AI41404 (United States NIAID NIH HHS)  
AI43198 (United States NIAID NIH HHS)  
AI45466 (United States NIAID NIH HHS)  
SO RNA (New York, N.Y.), (2003 Sep) Vol. 9, No. 9, pp. 1034-48.  
Journal code: 9509184. ISSN: 1355-8382.  
Report No.: NLM-PMC1370469.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
LA English  
FS Priority Journals  
EM 200310  
ED Entered STN: 23 Sep 2003  
Last Updated on STN: 17 Oct 2003  
Entered Medline: 16 Oct 2003  
AB Various chemical modifications were created in short-interfering RNAs ( siRNAs) to determine the biochemical properties required for RNA interference (RNAi). Remarkably, modifications at the 2'-position of pentose sugars in siRNAs showed the 2'-OHs were not required for RNAi, indicating that RNAi machinery does not require the 2'-OH for recognition of siRNAs and catalytic ribonuclease activity of RNA-induced silencing complexes (RISCs) does not involve the 2'-OH of guide antisense RNA. In addition, 2' modifications predicted to stabilize siRNA increased the persistence of RNAi as compared with wild-type siRNAs. RNAi was also induced with chemical modifications that stabilized interactions between A-U base pairs, demonstrating that these types of modifications may enhance mRNA targeting efficiency in

allele-specific RNAi. Modifications altering the structure of the A-form major groove of antisense siRNA-mRNA duplexes abolished RNAi, suggesting that the major groove of these duplexes was required for recognition by activated RISC\*. Comparative analysis of the stability and RNAi activities of chemically modified single-stranded antisense RNA and duplex siRNA suggested that some catalytic mechanism(s) other than siRNA stability were linked to RNAi efficiency. Modified or mismatched ribonucleotides incorporated at internal positions in the 5' or 3' half of the siRNA duplex, as defined by the antisense strand, indicated that the integrity of the 5' and not the 3' half of the siRNA structure was important for RNAi, highlighting the asymmetric nature of siRNA recognition for initiation of unwinding. Collectively, this study defines the mechanisms of RNAi in human cells and provides new rules for designing effective and stable siRNAs for RNAi-mediated gene-silencing applications.

L1 ANSWER 111 OF 117 MEDLINE on STN  
AN 2003463345 MEDLINE <<LOGINID::20090820>>  
DN PubMed ID: 12928480  
TI Allele-specific silencing of a pathogenic mutant acetylcholine receptor subunit by RNA interference.  
AU Abdelgany Amr; Wood Matthew; Beeson David  
CS Neurosciences Group, Weatherall Institute of Molecular Medicine, University of Oxford, UK.  
SO Human molecular genetics, (2003 Oct 15) Vol. 12, No. 20, pp. 2637-44.  
Electronic Publication: 2003-08-19.  
Journal code: 9208958. ISSN: 0964-6906.  
CY England: United Kingdom  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
LA English  
FS Priority Journals  
EM 200407  
ED Entered STN: 4 Oct 2003  
Last Updated on STN: 21 Jul 2004  
Entered Medline: 20 Jul 2004  
AB Slow channel congenital myasthenic syndrome (SCCMS) is a disorder of the neuromuscular synapse caused by dominantly inherited missense mutations in genes that encode the muscle acetylcholine receptor (AChR) subunits. Here we investigate the potential of post-transcriptional gene silencing using RNA interference (RNAi) for the selective down-regulation of pathogenic mutant AChR. By transfection of both siRNA and shRNA into mammalian cells expressing wild-type or mutant AChR subunits, we show, using  $^{125}\text{I}$ -alpha-bungarotoxin binding and immunofluorescence to measure cell surface AChR expression, efficient discrimination between the silencing of alphaS226F AChR mutant RNA transcripts and the wild-type. In this model we find that selectivity between mutant and wild-type transcripts is optimized with the nucleotide mismatch at position 9 in the shRNA complementary sequence. We also find that allele-specific silencing using shRNA has comparable efficiency to that using siRNA, underlining the general potential of stable expression of shRNA molecules as a long term therapeutic approach for allele-specific silencing of mutant transcripts in dominant genetic disorders.

L1 ANSWER 106 OF 117 MEDLINE on STN  
AN 2004041559 MEDLINE <<LOGINID::20090820>>  
DN PubMed ID: 14741366  
TI Enhancement of RNAi activity by improved siRNA duplexes.  
AU Hohjoh Hirohiko  
CS National Institute of Neuroscience, NCNP, 4-1-1 Ogawahigashi, Kodaira,

SO Tokyo 187-8502, Japan.. hohkohn@ncnp.go.jp  
FEBS letters, (2004 Jan 16) Vol. 557, No. 1-3, pp. 193-8.  
Journal code: 0155157. ISSN: 0014-5793.

CY Netherlands

DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English

FS Priority Journals

EM 200404

ED Entered STN: 27 Jan 2004  
Last Updated on STN: 7 Apr 2004  
Entered Medline: 6 Apr 2004

AB RNA interference (RNAi) is a powerful tool for suppressing the expression of a gene of interest, in which 21-25 nucleotide short interfering RNA (siRNA) duplexes homologous to the silenced gene function as sequence-specific RNAi mediators. The present study shows that newly designed siRNA duplexes, 'fork-siRNA duplexes', whose sense-stranded siRNA elements carry one to four nucleotide mismatches at the 3'-ends against the antisense-stranded siRNA elements, can enhance RNAi activity over conventional siRNA duplexes in cultured mammalian cells.

L1 ANSWER 95 OF 117 MEDLINE on STN  
AN 2004426817 MEDLINE <<LOGINID::20090820>>  
DN PubMed ID: 15331225

TI Picking a winner: new mechanistic insights into the design of effective siRNAs.

AU Gong Delquin; Ferrell James E Jr  
CS Department of Biological Sciences, Stanford University, Stanford, CA 94305-5020, USA.

SO Trends in biotechnology, (2004 Sep) Vol. 22, No. 9, pp. 451-4.  
Journal code: 8310903. ISSN: 0167-7799.

CY England: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200503

ED Entered STN: 28 Aug 2004  
Last Updated on STN: 16 Mar 2005  
Entered Medline: 15 Mar 2005

AB Recent work has shown that the efficacy of a small interfering RNA (siRNA) for silencing gene expression is a function of how easy it is to unwind the siRNA from the 5'-antisense end. Based on these insights, one group has designed an algorithm that substantially improves the odds of picking an effective siRNA, and two groups have shown that 'forked' or 'frayed' siRNAs, which should be easier to unwind from the 5'-antisense end, are more effective than conventional siRNAs. These strategies represent important steps towards the rational design of effective siRNAs.

L1 ANSWER 86 OF 117 MEDLINE on STN  
AN 2005106737 MEDLINE <<LOGINID::20090820>>  
DN PubMed ID: 15737617

TI Influence of assembly of siRNA elements into RNA-induced silencing complex by fork-siRNA duplex carrying nucleotide mismatches at the 3'- or 5'-end of the sense-stranded siRNA element.

AU Ohnishi Yusuke; Tokunaga Katsushi; Hohjoh Hirohiko  
CS National Institute of Neuroscience, NCNP, 4-1-1 Ogawahigashi, Kodaira, Tokyo 187-8502, Japan.

SO Biochemical and biophysical research communications, (2005 Apr 8) Vol.

329, No. 2, pp. 516-21.  
Journal code: 0372516. ISSN: 0006-291X.

CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
LA English  
FS Priority Journals  
EM 200505  
ED Entered STN: 2 Mar 2005  
Last Updated on STN: 6 May 2005  
Entered Medline: 5 May 2005

AB RNA interference (RNAi) is a powerful method for suppressing the expression of a gene of interest, and can be induced by 21-25 nucleotide small interfering RNA (siRNA) duplexes homologous to the silenced gene, which function as sequence-specific RNAi mediators in RNA-induced silencing complexes (RISCs). In the previous study, it was shown that fork-siRNA duplexes, whose sense-stranded siRNA elements carried a few nucleotide mismatches at the 3'-ends against the antisense-stranded siRNA elements, could enhance RNAi activity more than conventional siRNA duplexes in cultured mammalian cells. In this study, we further characterized fork-siRNA duplexes using reporter plasmids carrying target sequences complementary to the sense- or antisense-stranded siRNA elements in the untranslated region of Renilla luciferase. The data presented here suggest that nucleotide mismatches at either the 3'- or 5'-end of the sense-stranded siRNA elements in fork-siRNA duplexes could influence assembly of not only the antisense-stranded siRNA elements but also the sense-stranded elements into RISCs. In addition, we further suggest the possibility that there could be a positional effect of siRNA duplex on RNAi activity.

L1 ANSWER 85 OF 117 MEDLINE on STN  
AN 2005121791 MEDLINE <>LOGINID::20090820>>  
DN PubMed ID: 15752771  
TI Evaluation of in vitro inhibitory potential of small interfering RNAs directed against various regions of foot-and-mouth disease virus genome.  
AU Mohapatra Jajati Keshari; Sanyal Aniket; Hemadri Divakar; Tosh Chakradhar; Kumar R Manoj; Bandyopadhyay Santanu Kumar  
CS Project Directorate on Foot-and-Mouth Disease, Indian Veterinary Research Institute Campus, Mukteswar-Kumaon, Nainital 263 138, Uttaranchal, India.  
SO Biochemical and biophysical research communications, (2005 Apr 15) Vol. 329, No. 3, pp. 1133-8.  
Journal code: 0372516. ISSN: 0006-291X.

CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
LA English  
FS Priority Journals  
EM 200505  
ED Entered STN: 9 Mar 2005  
Last Updated on STN: 17 May 2005  
Entered Medline: 16 May 2005

AB India is endemic for foot-and-mouth disease and it continues to be a major threat to the livestock industry despite vaccination programmes. In the present study, the ability of specific small interfering (si)RNAs directed against different genomic regions of foot-and-mouth disease virus (FMDV) to inhibit virus replication in BHK-21 cells was examined. For preliminary evaluation of possible siRNA-mediated FMDV inhibition, a cocktail of several unique populations of 12-30bp siRNAs were successfully produced corresponding to three target regions located at structural (VP3-VP1), non-structural (2A-2C), and

non-structural-untranslated (3D-3'UTR) region of serotype Asial. Once the populations of siRNAs generated were found to reduce the virus titre significantly, two highly conserved 21bp siRNA duplexes were designed by analysing all FMDV sequence entries available in public-domain databases. In virus titration assay, more than 99% inhibition of virus yield for all the four serotypes (type Asial, O, A, and C) could be demonstrated in cells transfected with each of the FMDV-specific siRNAs at 24h post-infection, compared to control cells transfected with scrambled siRNA. This was well supported by reduction in OD values in FMDV-specific sandwich ELISA. Although 100-fold reduction in virus titre with siRNA1 is substantial considering the transfection efficiency and fixed level of input siRNA, siRNA2 emerged to be a better choice as target where more than 300-fold reduction was observed and its inhibitory effect extended up to 48 h post-infection against all the serotypes. Interestingly, in the present study type A virus (IND 17/77) had a single mismatch at position 2 in the siRNA2 target region but it did not abrogate the inhibitory effect.

L1 ANSWER 84 OF 117 MEDLINE on STN  
AN 2005149865 MEDLINE <>LOGINID::20090820>>  
DN PubMed ID: 15781493  
TI A systematic analysis of the silencing effects of an active siRNA at all single-nucleotide mismatched target sites.  
AU Du Quan; Thonberg Hakan; Wang Jue; Wahlestedt Claes; Liang Zicai  
CS Center for Genomics and Bioinformatics, Karolinska Institutet 171 77 Stockholm, Sweden.. quan.du@cgb.ki.se  
SO Nucleic acids research, (2005) Vol. 33, No. 5, pp. 1671-7. Electronic Publication: 2005-03-21.  
Journal code: 0411011. E-ISSN: 1362-4962.  
Report No.: NLM-PMC1069010.  
CY England: United Kingdom  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
LA English  
FS Priority Journals  
EM 200503  
ED Entered STN: 23 Mar 2005  
Last Updated on STN: 29 Mar 2005  
Entered Medline: 28 Mar 2005  
AB The specificity of small interfering RNA (siRNA)-mediated gene silencing is a critical consideration for the application of RNA interference (RNAi). While the discovery of potential off-target effects by siRNAs is of concern, no systematic analysis has been conducted to explore the specificity of RNAi. Here, we present a study where a functionally validated siRNA (siCD46) was examined for silencing specificity on all possible 57 permuted target sites, each carrying a single-nucleotide mutation that would generate a mismatch when paired with siRNA antisense strand. We found that it was not only the position of the mismatched base pair, but also the identity of the nucleotides forming the mismatch that influenced silencing. Surprisingly, mismatches formed between adenine (A) and cytosine (C), in addition to the G:U wobble base pair, were well tolerated and target sites containing such mismatches were silenced almost as efficiently as its fully matched counterpart by siCD46. Northern blots showed that the silencing of fusion genes harboring the mutated target sites involved target mRNA degradation. This study provides direct evidence that the target recognition of siRNA is far more degenerative than previously considered. This finding is instrumental in the understanding of RNAi specificity and may aid the computational prediction of RNA

secondary structure.

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=> d his full

(FILE 'HOME' ENTERED AT 07:14:07 ON 20 AUG 2009)

FILE 'MEDLINE' ENTERED AT 07:14:20 ON 20 AUG 2009  
L1 117 SEA PLU=ON MISMATCH? AND SIRNA  
D TI 70-117  
D BIB AB 114 116 112 111 106 95 86 85 84

FILE 'STNGUIDE' ENTERED AT 07:17:51 ON 20 AUG 2009

FILE HOME

FILE MEDLINE

FILE LAST UPDATED: 19 Aug 2009 (20090819/UP). FILE COVERS 1949 TO DATE.

MEDLINE and LMEDLINE have been updated with the 2009 Medical Subject Headings (MeSH) vocabulary and tree numbers from the U.S. National Library of Medicine (NLM). Additional information is available at

[http://www.nlm.nih.gov/pubs/techbull/nd08/nd08\\_medline\\_data\\_changes\\_2009](http://www.nlm.nih.gov/pubs/techbull/nd08/nd08_medline_data_changes_2009).

On February 21, 2009, MEDLINE was reloaded. See HELP RLOAD for details.

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See HELP RANGE before carrying out any RANGE search.

FILE STNGUIDE  
FILE CONTAINS CURRENT INFORMATION.  
LAST RELOADED: Aug 14, 2009 (20090814/UP).

=> d ti 40-69  
YOU HAVE REQUESTED DATA FROM FILE 'MEDLINE' - CONTINUE? (Y)/N:y

L1 ANSWER 40 OF 117 MEDLINE on STN  
TI RFRCDB-siRNA: improved design of siRNAs by random forest regression model coupled with database searching.

L1 ANSWER 41 OF 117 MEDLINE on STN  
TI Drosophila microRNAs are sorted into functionally distinct argonaute

complexes after production by dicer-1.

L1 ANSWER 42 OF 117 MEDLINE on STN  
TI BRCA1 activates a G2-M cell cycle checkpoint following 6-thioguanine-induced DNA mismatch damage.

L1 ANSWER 43 OF 117 MEDLINE on STN  
TI Novel insights into RNAi off-target effects using *C. elegans* paralogs.

L1 ANSWER 44 OF 117 MEDLINE on STN  
TI CPT-11 (SN-38) chemotherapy may be selectively applicable to biliary tract cancer with low hMLH1 expression.

L1 ANSWER 45 OF 117 MEDLINE on STN  
TI Inhibition of multiple strains of Venezuelan equine encephalitis virus by a pool of four short interfering RNAs.

L1 ANSWER 46 OF 117 MEDLINE on STN  
TI Implications of phase transitions in knockdown networks of transitive RNAi.

L1 ANSWER 47 OF 117 MEDLINE on STN  
TI Arrest of cancer cell proliferation by dsRNAs.

L1 ANSWER 48 OF 117 MEDLINE on STN  
TI Comparison of siRNA-induced off-target RNA and protein effects.

L1 ANSWER 49 OF 117 MEDLINE on STN  
TI Inhibition of SARS-CoV replication cycle by small interference RNAs silencing specific SARS proteins, 7a/7b, 3a/3b and S.

L1 ANSWER 50 OF 117 MEDLINE on STN  
TI Small interfering RNAs targeting mutant K-ras inhibit human pancreatic carcinoma cells growth in vitro and in vivo.

L1 ANSWER 51 OF 117 MEDLINE on STN  
TI Secondary siRNAs result from unprimed RNA synthesis and form a distinct class.

L1 ANSWER 52 OF 117 MEDLINE on STN  
TI Gene silencing activity of siRNAs with a ribo-difluorotoluyl nucleotide.

L1 ANSWER 53 OF 117 MEDLINE on STN  
TI Designing siRNA that distinguish between genes that differ by a single nucleotide.

L1 ANSWER 54 OF 117 MEDLINE on STN  
TI Expression of vector-based small interfering RNA against West Nile virus effectively inhibits virus replication.

L1 ANSWER 55 OF 117 MEDLINE on STN  
TI RiboSubstrates: a web application addressing the cleavage specificities of ribozymes in designated genomes.

L1 ANSWER 56 OF 117 MEDLINE on STN  
TI RNA interference in vitro and in vivo using a novel chitosan/siRNA nanoparticle system.

L1 ANSWER 57 OF 117 MEDLINE on STN  
TI DNA mismatch repair as an effector for promoting phorbol

ester-induced apoptotic DNA damage and cell killing: implications in tumor promotion.

L1 ANSWER 58 OF 117 MEDLINE on STN  
TI Widespread siRNA "off-target" transcript silencing mediated by seed region sequence complementarity.

L1 ANSWER 59 OF 117 MEDLINE on STN  
TI Mismatched siRNAs downregulate mRNAs as a function of target site location.

L1 ANSWER 60 OF 117 MEDLINE on STN  
TI XPA versus ERCC1 as chemosensitising agents to cisplatin and mitomycin C in prostate cancer cells: role of ERCC1 in homologous recombination repair.

L1 ANSWER 61 OF 117 MEDLINE on STN  
TI Antiviral activity of small interfering RNAs: specificity testing using heterologous virus reveals interferon-related effects overlooked by conventional mismatch controls.

L1 ANSWER 62 OF 117 MEDLINE on STN  
TI Mammary gland tissue targeted overexpression of human protease-activated receptor 1 reveals a novel link to beta-catenin stabilization.

L1 ANSWER 63 OF 117 MEDLINE on STN  
TI Improved targeting of miRNA with antisense oligonucleotides.

L1 ANSWER 64 OF 117 MEDLINE on STN  
TI Determinants of specific RNA interference-mediated silencing of human beta-globin alleles differing by a single nucleotide polymorphism.

L1 ANSWER 65 OF 117 MEDLINE on STN  
TI 3' UTR seed matches, but not overall identity, are associated with RNAi off-targets.

L1 ANSWER 66 OF 117 MEDLINE on STN  
TI Hypoxia-inducible factor-1alpha polymorphisms and TSC1/2 mutations are complementary in head and neck cancers.

L1 ANSWER 67 OF 117 MEDLINE on STN  
TI Transcription promotes contraction of CAG repeat tracts in human cells.

L1 ANSWER 68 OF 117 MEDLINE on STN  
TI Transgenic small interfering RNA halts amyotrophic lateral sclerosis in a mouse model.

L1 ANSWER 69 OF 117 MEDLINE on STN  
TI Inhibition of drug-resistant HIV-1 by RNA interference.

=> d bib ab 59  
YOU HAVE REQUESTED DATA FROM FILE 'MEDLINE' - CONTINUE? (Y)/N:y

L1 ANSWER 59 OF 117 MEDLINE on STN  
AN 2006369956 MEDLINE <<LOGINID::20090820>>  
DN PubMed ID: 16764866  
TI Mismatched siRNAs downregulate mRNAs as a function of

AU target site location.  
AU Martin Scott E; Caplen Natasha J  
CS Gene Silencing Section, Office of Science and Technology Partnerships,  
Office of the Director, CCR, NCI, NIH, Bethesda, MD 20892, USA.  
SO FEBS letters, (2006 Jun 26) Vol. 580, No. 15, pp. 3694-8. Electronic  
Publication: 2006-06-05.  
Journal code: 0155157. ISSN: 0014-5793.  
CY Netherlands  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, N.I.H., INTRAMURAL)  
LA English  
FS Priority Journals  
EM 200608  
ED Entered STN: 21 Jun 2006  
Last Updated on STN: 9 Aug 2006  
Entered Medline: 8 Aug 2006  
AB In mammalian cells, RNA interference can be mediated by synthetic duplex  
RNAs, termed small interfering RNAs (siRNAs), which assist in  
cleaving completely complementary mRNA transcripts. MicroRNAs (miRNAs)  
are endogenous small RNAs that assist in translationally repressing mRNAs  
with regions of partial complementarity, but may also reduce transcript  
levels. Since miRNAs predominantly interact with the 3' UTRs of  
transcripts, we sought to ask if mismatched siRNAs  
mimicking miRNAs affect cognate mRNA levels as a function of target site  
location. We find that mismatched siRNAs targeting  
the 3' UTRs of two endogenous transcripts yield a greater reduction in  
mRNA levels than those targeting the coding region. Our findings  
demonstrate the importance of target site location within endogenous mRNAs  
for small RNAs associated with RNAi.

=> d his full

(FILE 'HOME' ENTERED AT 07:14:07 ON 20 AUG 2009)

FILE 'MEDLINE' ENTERED AT 07:14:20 ON 20 AUG 2009  
L1 117 SEA PLU=ON MISMATCH? AND SIRNA  
D TI 70-117  
D BIB AB 114 116 112 111 106 95 86 85 84

FILE 'STNGUIDE' ENTERED AT 07:17:51 ON 20 AUG 2009

FILE 'MEDLINE' ENTERED AT 07:31:23 ON 20 AUG 2009  
D TI 40-69

FILE 'STNGUIDE' ENTERED AT 07:31:24 ON 20 AUG 2009

FILE 'MEDLINE' ENTERED AT 07:32:56 ON 20 AUG 2009  
D BIB AB 59

FILE 'STNGUIDE' ENTERED AT 07:32:56 ON 20 AUG 2009

FILE HOME

FILE MEDLINE

FILE LAST UPDATED: 19 Aug 2009 (20090819/UP). FILE COVERS 1949 TO DATE.

MEDLINE and LMEDLINE have been updated with the 2009 Medical Subject  
Headings (MeSH) vocabulary and tree numbers from the U.S. National Library  
of Medicine (NLM). Additional information is available at

[http://www.nlm.nih.gov/pubs/techbull/nd08/nd08\\_medline\\_data\\_changes\\_2009.html](http://www.nlm.nih.gov/pubs/techbull/nd08/nd08_medline_data_changes_2009.html)

On February 21, 2009, MEDLINE was reloaded. See HELP RLOAD for details.

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FILE STNGUIDE  
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FILE 'MEDLINE' ENTERED AT 08:23:33 ON 20 AUG 2009

FILE LAST UPDATED: 19 Aug 2009 (20090819/UP). FILE COVERS 1949 TO DATE.

MEDLINE and LMEDLINE have been updated with the 2009 Medical Subject Headings (MeSH) vocabulary and tree numbers from the U.S. National Library of Medicine (NLM). Additional information is available at

[http://www.nlm.nih.gov/pubs/techbull/nd08/nd08\\_medline\\_data\\_changes\\_2009.html](http://www.nlm.nih.gov/pubs/techbull/nd08/nd08_medline_data_changes_2009.html).

On February 21, 2009, MEDLINE was reloaded. See HELP RLOAD for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

See HELP RANGE before carrying out any RANGE search.

=> s dicer and substrate and mismatch?  
758 DICER  
19 DICERS  
761 DICER

(DICER OR DICERS)  
230111 SUBSTRATE  
103276 SUBSTRATES  
289475 SUBSTRATE  
(SUBSTRATE OR SUBSTRATES)  
26652 MISMATCH?  
L1 1 DICER AND SUBSTRATE AND MISMATCH?  
  
=> d bib ab

L1 ANSWER 1 OF 1 MEDLINE on STN  
AN 2008647339 MEDLINE <<LOGINID::20090820>>  
DN PubMed ID: 18839783  
TI Inhibition of human metapneumovirus replication by small interfering RNA.  
AU Deffrasnes Celine; Cavanagh Marie-Helene; Goyette Nathalie; Cui Kunyuan;  
Ge Qing; Seth Shaguna; Templin Michael V; Quay Steven C; Johnson Paul H;  
Boivin Guy  
CS Infectious Disease Research Centre of the Centre Hospitalier Universitaire  
de Quebec, Laval University, Quebec City, QC, Canada.  
SO Antiviral therapy, (2008) Vol. 13, No. 6, pp. 821-32.  
Journal code: 9815705. ISSN: 1359-6535.  
CY England: United Kingdom  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
LA English  
FS Priority Journals  
EM 200811  
ED Entered STN: 9 Oct 2008  
Last Updated on STN: 8 Nov 2008  
Entered Medline: 7 Nov 2008  
AB BACKGROUND: Human metapneumovirus (hMPV) is a major respiratory viral pathogen in young children, elderly individuals and immunocompromised patients. Despite its major effects related to bronchiolitis, pneumonia and its potential role in recurrent wheezing episodes, there is still no commercial treatment or vaccine available against this paramyxovirus. METHODS: We tested a therapeutic strategy for hMPV that was based on RNA interference. RESULTS: An hMPV genome-wide search for small interfering RNAs (siRNAs) by computational analysis revealed 200 potentially effective 21-mer siRNAs. Initial screening with a luciferase assay identified 57 siRNAs of interest. Further evaluation of their inhibitory potential against the four hMPV subgroups by quantitative real-time reverse transcriptase PCR and plaque immunoassay identified two highly potent siRNAs with 50% inhibitory concentration (IC50) values in the subnanomolar range. siRNA45 targets the nucleoprotein messenger RNA (mRNA) and had IC50 values <0.078 nM against representative strains from the four hMPV subgroups, whereas siRNA60, which targets the phosphoprotein mRNA, had IC50 values between 0.090-<0.078 nM against the same panel of hMPV strains. Longer 25/27-mer siRNAs known as Dicer substrates designed from the top two siRNA candidates were also evaluated and were at least as effective as their corresponding 21-mer siRNAs. Interestingly, the presence of one or two nucleotide mismatches in the target mRNA sequence of some hMPV subgroups did not always affect hMPV inhibition in vitro. CONCLUSIONS: We successfully identified two highly efficient siRNAs against hMPV targeting essential components of the hMPV replication complex.

=> s dicer and mismatch?  
758 DICER  
19 DICERS  
761 DICER

(DICER OR DICERS)  
26652 MISMATCH?  
L2 9 DICER AND MISMATCH?

=> d ti 1-9

L2 ANSWER 1 OF 9 MEDLINE on STN  
TI Inhibition of human metapneumovirus replication by small interfering RNA.

L2 ANSWER 2 OF 9 MEDLINE on STN  
TI Two molecular features contribute to the Argonaute specificity for the microRNA and RNAi pathways in *C. elegans*.

L2 ANSWER 3 OF 9 MEDLINE on STN  
TI MicroRNA-373 induces expression of genes with complementary promoter sequences.

L2 ANSWER 4 OF 9 MEDLINE on STN  
TI Endoribonuclease-prepared short interfering RNAs induce effective and specific inhibition of human immunodeficiency virus type 1 replication.

L2 ANSWER 5 OF 9 MEDLINE on STN  
TI Drosophila microRNAs are sorted into functionally distinct argonaute complexes after production by dicer-1.

L2 ANSWER 6 OF 9 MEDLINE on STN  
TI Prevalence of off-target effects in Drosophila RNA interference screens.

L2 ANSWER 7 OF 9 MEDLINE on STN  
TI A computational study of off-target effects of RNA interference.

L2 ANSWER 8 OF 9 MEDLINE on STN  
TI Derivation and function of small interfering RNAs and microRNAs.

L2 ANSWER 9 OF 9 MEDLINE on STN  
TI Functional siRNAs and miRNAs exhibit strand bias.

=> s dicer and substrate  
758 DICER  
19 DICERS  
761 DICER  
(DICER OR DICERS)  
230111 SUBSTRATE  
103276 SUBSTRATES  
289475 SUBSTRATE  
(SUBSTRATE OR SUBSTRATES)

L3 89 DICER AND SUBSTRATE

=> d ti 50-89

L3 ANSWER 50 OF 89 MEDLINE on STN  
TI Reliable prediction of Drosha processing sites improves microRNA gene prediction.

L3 ANSWER 51 OF 89 MEDLINE on STN  
TI RNA interference from multimeric shRNAs generated by rolling circle transcription.

L3 ANSWER 52 OF 89 MEDLINE on STN  
TI Characterization of the short RNAs bound by the P19 suppressor of RNA

silencing in mouse embryonic stem cells.

L3 ANSWER 53 OF 89 MEDLINE on STN  
TI Sequestration and protection of double-stranded RNA by the betanodavirus b2 protein.

L3 ANSWER 54 OF 89 MEDLINE on STN  
TI Induction of the interferon response by siRNA is cell type- and duplex length-dependent.

L3 ANSWER 55 OF 89 MEDLINE on STN  
TI A structural basis for discriminating between self and nonself double-stranded RNAs in mammalian cells.

L3 ANSWER 56 OF 89 MEDLINE on STN  
TI Characterization of 43 non-protein-coding mRNA genes in Arabidopsis, including the MIR162a-derived transcripts.

L3 ANSWER 57 OF 89 MEDLINE on STN  
TI Molecular phylogenetics and comparative modeling of HEN1, a methyltransferase involved in plant microRNA biogenesis.

L3 ANSWER 58 OF 89 MEDLINE on STN  
TI Short interfering RNA strand selection is independent of dsRNA processing polarity during RNAi in Drosophila.

L3 ANSWER 59 OF 89 MEDLINE on STN  
TI Mammalian microRNAs: a small world for fine-tuning gene expression.

L3 ANSWER 60 OF 89 MEDLINE on STN  
TI Sequence-specific interference by small RNAs derived from adenovirus VAI RNA.

L3 ANSWER 61 OF 89 MEDLINE on STN  
TI Structural insight into the mechanism of double-stranded RNA processing by ribonuclease III.

L3 ANSWER 62 OF 89 MEDLINE on STN  
TI Functional proteomics reveals the biochemical niche of C. elegans DCR-1 in multiple small-RNA-mediated pathways.

L3 ANSWER 63 OF 89 MEDLINE on STN  
TI Structural basis for double-stranded RNA processing by Dicer.

L3 ANSWER 64 OF 89 MEDLINE on STN  
TI A human, ATP-independent, RISC assembly machine fueled by pre-miRNA.

L3 ANSWER 65 OF 89 MEDLINE on STN  
TI Human RISC couples microRNA biogenesis and posttranscriptional gene silencing.

L3 ANSWER 66 OF 89 MEDLINE on STN  
TI Approaches for chemically synthesized siRNA and vector-mediated RNAi.

L3 ANSWER 67 OF 89 MEDLINE on STN  
TI Control of peptide product sizes by the energy-dependent protease ClpAP.

L3 ANSWER 68 OF 89 MEDLINE on STN  
TI Extensive variation in the 5'-UTR of Dicer mRNAs influences translational efficiency.

L3 ANSWER 69 OF 89 MEDLINE on STN  
TI Functional polarity is introduced by Dicer processing of short substrate RNAs.

L3 ANSWER 70 OF 89 MEDLINE on STN  
TI Dicer-1 and R3D1-L catalyze microRNA maturation in Drosophila.

L3 ANSWER 71 OF 89 MEDLINE on STN  
TI Suppression of RNA interference by adenovirus virus-associated RNA.

L3 ANSWER 72 OF 89 MEDLINE on STN  
TI Natural antisense transcripts with coding capacity in Arabidopsis may have a regulatory role that is not linked to double-stranded RNA degradation.

L3 ANSWER 73 OF 89 MEDLINE on STN  
TI A virus-encoded inhibitor that blocks RNA interference in mammalian cells.

L3 ANSWER 74 OF 89 MEDLINE on STN  
TI Identification of a peach latent mosaic viroid hairpin able to act as a Dicer-like substrate.

L3 ANSWER 75 OF 89 MEDLINE on STN  
TI Complete, gene-specific siRNA libraries: production and expression in mammalian cells.

L3 ANSWER 76 OF 89 MEDLINE on STN  
TI The contributions of dsRNA structure to Dicer specificity and efficiency.

L3 ANSWER 77 OF 89 MEDLINE on STN  
TI Catalytic mechanism of Escherichia coli ribonuclease III: kinetic and inhibitor evidence for the involvement of two magnesium ions in RNA phosphodiester hydrolysis.

L3 ANSWER 78 OF 89 MEDLINE on STN  
TI Synthetic dsRNA Dicer substrates enhance RNAi potency and efficacy.

L3 ANSWER 79 OF 89 MEDLINE on STN  
TI Synthetic shRNAs as potent RNAi triggers.

L3 ANSWER 80 OF 89 MEDLINE on STN  
TI RNA interference and its promising future.

L3 ANSWER 81 OF 89 MEDLINE on STN  
TI Testis brain ribonucleic acid-binding protein/translin possesses both single-stranded and double-stranded ribonuclease activities.

L3 ANSWER 82 OF 89 MEDLINE on STN  
TI Structural basis for overhang-specific small interfering RNA recognition by the PAZ domain.

L3 ANSWER 83 OF 89 MEDLINE on STN  
TI The fragile X syndrome repeats form RNA hairpins that do not activate the interferon-inducible protein kinase, PKR, but are cut by Dicer.

L3 ANSWER 84 OF 89 MEDLINE on STN  
TI Human Dicer preferentially cleaves dsRNAs at their termini without a requirement for ATP.

L3 ANSWER 85 OF 89 MEDLINE on STN

TI MicroRNA maturation: stepwise processing and subcellular localization.

L3 ANSWER 86 OF 89 MEDLINE on STN

TI The dsRNA binding protein RDE-4 interacts with RDE-1, DCR-1, and a DExH-box helicase to direct RNAi in *C. elegans*.

L3 ANSWER 87 OF 89 MEDLINE on STN

TI Short hairpin RNAs (shRNAs) induce sequence-specific silencing in mammalian cells.

L3 ANSWER 88 OF 89 MEDLINE on STN

TI Dicer functions in RNA interference and in synthesis of small RNA involved in developmental timing in *C. elegans*.

L3 ANSWER 89 OF 89 MEDLINE on STN

TI Role for a bidentate ribonuclease in the initiation step of RNA interference.

=> d bib ab 78 76 63

L3 ANSWER 78 OF 89 MEDLINE on STN

AN 2005067203 MEDLINE <<LOGINID::20090820>>

DN PubMed ID: 15619617

TI Synthetic dsRNA Dicer substrates enhance RNAi potency and efficacy.

AU Kim Dong-Ho; Behlke Mark A; Rose Scott D; Chang Mi-Sook; Choi Sangdun; Rossi John J

NC AI29329 (United States NIAID NIH HHS)

AI42552 (United States NIAID NIH HHS)

HL074704 (United States NHLBI NIH HHS)

SO Nature biotechnology, (2005 Feb) Vol. 23, No. 2, pp. 222-6. Electronic Publication: 2004-12-26.

Journal code: 9604648. ISSN: 1087-0156.

CY United States

DT Letter

(RESEARCH SUPPORT, NON-U.S. GOV'T)

(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)

LA English

FS Priority Journals

EM 200505

ED Entered STN: 8 Feb 2005

Last Updated on STN: 1 Jun 2005

Entered Medline: 31 May 2005

AB RNA interference (RNAi) is the process of sequence-specific post-transcriptional gene silencing triggered by double-stranded RNAs. In attempts to identify RNAi triggers that effectively function at lower concentrations, we found that synthetic RNA duplexes 25-30 nucleotides in length can be up to 100-fold more potent than corresponding conventional 21-mer small interfering RNAs (siRNAs). Some sites that are refractory to silencing by 21-mer siRNAs can be effectively targeted by 27-mer duplexes, with silencing lasting up to 10 d. Notably, the 27-mers do not induce interferon or activate protein kinase R (PKR). The enhanced potency of the longer duplexes is attributed to the fact that they are substrates of the Dicer endonuclease, directly linking the production of siRNAs to incorporation in the RNA-induced silencing complex. These results provide an alternative strategy for eliciting RNAi-mediated target cleavage using low concentrations of synthetic RNA as substrates for cellular Dicer-mediated cleavage.

L3 ANSWER 76 OF 89 MEDLINE on STN

AN 2005206825 MEDLINE <<LOGINID::20090820>>  
DN PubMed ID: 15811921  
TI The contributions of dsRNA structure to Dicer specificity and efficiency.  
AU Vermeulen Annaleen; Behlen Linda; Reynolds Angela; Wolfson Alexey;  
Marshall William S; Karpilow Jon; Khvorova Anastasia  
CS Dharmacon Inc., 2650 Crescent Dr., Suite #100, Lafayette, CO 80026, USA.  
SO RNA (New York, N.Y.), (2005 May) Vol. 11, No. 5, pp. 674-82. Electronic Publication: 2005-04-05.  
Journal code: 9509184. ISSN: 1355-8382.  
Report No.: NLM-PMC1370754.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)  
LA English  
FS Priority Journals  
EM 200505  
ED Entered STN: 21 Apr 2005  
Last Updated on STN: 25 May 2005  
Entered Medline: 24 May 2005  
AB Dicer processes long double-stranded RNA (dsRNA) and pre-microRNAs to generate the functional intermediates (short interfering RNAs and microRNAs) of the RNA interference pathway. Here we identify features of RNA structure that affect Dicer specificity and efficiency. The data presented show that various attributes of the 3' end structure, including overhang length and sequence composition, play a primary role in determining the position of Dicer cleavage in both dsRNA and unimolecular, short hairpin RNA (shRNA). We also demonstrate that siRNA end structure affects overall silencing functionality. Awareness of these new features of Dicer cleavage specificity as it is related to siRNA functionality provides a more detailed understanding of the RNAi mechanism and can shape the development of hairpins with enhanced functionality.

L3 ANSWER 63 OF 89 MEDLINE on STN  
AN 2006024015 MEDLINE <<LOGINID::20090820>>  
DN PubMed ID: 16410517  
TI Structural basis for double-stranded RNA processing by Dicer.  
AU Macrae Ian J; Zhou Kaihong; Li Fei; Repic Adrian; Brooks Angela N; Cande W Zacheus; Adams Paul D; Doudna Jennifer A  
CS Department of Molecular and Cell Biology, University of California, Berkeley, CA 94720, USA.  
SO Science (New York, N.Y.), (2006 Jan 13) Vol. 311, No. 5758, pp. 195-8.  
Journal code: 0404511. E-ISSN: 1095-9203.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
LA English  
FS Priority Journals  
OS PDB-2FFL  
EM 200602  
ED Entered STN: 14 Jan 2006  
Last Updated on STN: 2 Feb 2006  
Entered Medline: 1 Feb 2006  
AB The specialized ribonuclease Dicer initiates RNA interference by cleaving double-stranded RNA (dsRNA) substrates into small fragments about 25 nucleotides in length. In the crystal structure of an intact Dicer enzyme, the PAZ domain, a module that binds the end of dsRNA, is separated from the two catalytic ribonuclease III (RNase III) domains by a flat, positively charged surface. The 65 angstrom distance

between the PAZ and RNase III domains matches the length spanned by 25 base pairs of RNA. Thus, Dicer itself is a molecular ruler that recognizes dsRNA and cleaves a specified distance from the helical end.

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L1 1 SEA PLU=ON DICER AND SUBSTRATE AND MISMATCH?  
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L2 9 SEA PLU=ON DICER AND MISMATCH?  
D TI 1-9  
L3 89 SEA PLU=ON DICER AND SUBSTRATE  
D TI 50-89  
D BIB AB 78 76 63

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